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A WITHIN-SUBJECT ANALYSIS OF STIMULUS CONTROL THERAPY WITH SEVERE SLEEP-ONSET INSOMNIA

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Summary—Stimulus control, a behavioral technique designed to reduce sleep difficulties, has been demonstrated to be effective when compared with control procedures. These comparisons, mainly involving between-subjects analyses, have neglected the contribution of the stimulus control procedure to the production of clinically significant amelioration of sleep dysfunction. In contrast, the present within-subjects experiment was conducted to assess the capability of stimulus control to produce clinically relevant reductions in multiple measures of sleep disturbance. A comparison with the credible placebo procedure indicated that the stimulus control techniques reduced subjects' sleep onset latency to a mean latency below 30 min per week. Additionally, sedative–hypnotic usage was greatly reduced.

Chemotherapy represents the most common treatment for sleep disorders (Williams and Karacan, 1976; AMA Department of Drugs, 1977). This popularity is due mainly to the relatively rapid, if transient, relief of sleep-related complaints resulting from the use of sedative–hypnotic drugs. Unfortunately, these substances have been implicated in the production of a number of deleterious physiological and psychological effects calling their continued high frequency of use into question (AMA Department of Drugs, 1977; Williams and Karacan, 1976). In an effort to develop a rapid method of ameliorating sleep dysfunction devoid of the negative aspects of chemotherapy, a number of behavior therapists have suggested and, in some cases, demonstrated the effectiveness of various behavioral approaches to the problem (Ascher and Efran, 1978; Borkovec and Fowles, 1973; Borkovec *et al.*, 1975; Borkovec *et al.*, 1973; Haynes *et al.*, 1975; Nicassio and Bootzin, 1974; Steinmark and Borkovec, 1974; Woolfolk *et al.*, 1976; Lawrence and Tokarz, 1976; Slama, 1975; Tokarz and Lawrence, 1974; Turner and Ascher, 1978; Zwart and Lisman, 1976). Among these is a procedure-labeled stimulus control. While the concept of stimulus control was first developed by Brown (1965) in the context of infra-human operant conditioning, it was utilized by Bootzin (1972) as the rationale for a clinical procedure designed to treat sleep disorders. Several experimental investigations have been generated for the purpose of examining Bootzin's (1972) proposal (Bootzin and Nicassio, in press).

The data from these investigations (Bootzin, 1975; Haynes *et al.*, 1975; Lawrence and Tokarz, 1976; Tokarz and Lawrence, 1974; Slama, 1975; Turner and Ascher, 1978) have clearly indicated that stimulus control therapy is more effective than no treatment and placebo conditions for the treatment of sleep disturbance.

With the exception of the Haynes *et al.* (1975) study, the investigations of stimulus control therapy have been between groups studies. Unfortunately, this type of design fails to adequately assess the clinical significance of a proposed therapeutic procedure (Hersen and Barlow, 1976). In their attempt to study the clinical efficacy of stimulus control therapy, Haynes *et al.* (1975) utilized an A–B–A–B design, but failed to obtain an unambiguous reversal effect of a sufficient magnitude to warrant a cause–effect statement. In fact, the authors reported that two of the four clients failed to show any sign of reversal movement. This finding is not surprising since, as Hersen and Barlow (1976), among others, have suggested, many behavioral treatments involve a carry-over

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effect. The use of a reversal design (e.g. of the A–B–A–B type) is therefore ill-advised with such behavioral procedures because reproduction of the baseline condition (i.e. a cessation of the treatment) will probably not result in the client returning to basal behavior. In such cases, a cause–effect relationship can more adequately be demonstrated through a multiple baseline design (Hersen and Barlow, 1976).

The purpose of the present study is that of testing the clinical efficacy of the stimulus control procedure by utilizing a design (i.e. a multiple baseline across subjects) which can clearly support a cause–effect statement. This represents data which is complementary to that of the several studies which have demonstrated the statistical efficacy of this treatment method. Monroe (1967) has defined sleep onset insomnia as greater than a 30 min latency to sleep onset on three or more nights per week. For this purpose of the present paper an individual will be assumed to have achieved a clinically significant level of improvement when his average sleep onset latency is less than 30 min per week, and further therapy for this problem is precluded. While sleep onset latency defines the major dependent variable for the present study, other measures of sleep disturbance will also be monitored.

METHOD

Subjects

Clients were self-referred outpatients seen in the clinic of the Behavior Therapy Unit of Temple University Medical School; each presented with the primary complaint of insomnia. Six clients who exhibited greater than a weekly mean of 60 min latency to sleep onset were utilized for the present study.

Client 1. A 61-year-old male, presenting a 25-year history of sleep dysfunction and a 15-year history of constant usage of sleep medication which, at the time of treatment, consisted of 500 mg of glutethimide and 25 mg of diazepam per night. Upon the initiation of therapy the client was taking 107 min to fall asleep on the average.

Client 2. A 32-year-old female, presenting a 20-year history of sleep disturbance. At the beginning of therapy she was taking 400 mg of amobarbital per night and required 91 min to fall asleep.

Client 3. A 60-year-old male having experienced sleep dysfunction for 15 years. He was using 200 mg of secobarbital per night, yet averaging 84 min latency to sleep onset.

Client 4. A 46-year-old female with a 10-year history of sleep dysfunction reported using 250 mg of glutethimide each evening and requiring 113 min to fall asleep.

Client 5. A 49-year-old female with a 12-year history of sleep disturbance. She habitually took 30 mg of flurazepam per night and required 66 min to fall asleep.

Client 6. A 34-year-old male who experienced sleep disturbance for a period of 8 years. Although the client was taking no sleep medication, *per se*, he was drinking two cans of beer prior to bedtime in an effort to fall asleep; notwithstanding, he reported a mean latency of 90 min to sleep onset.

Design

During the first week all clients participated in a baseline phase. During weeks 2–5 clients 1, 2 and 3 received stimulus control therapy (a condition in which they were maintained for the duration of the study), while clients 4, 5 and 6 received placebo therapy. Beginning with week 6 clients 4, 5 and 6 were switched to stimulus control therapy to the conclusion of the study.

Measures

The primary dependent variables consisted of the subjects' reports of the number of minutes to sleep onset and the type and amount of sleep-inducing substance taken per night. In addition the following ancillary measures of clients' sleep quality were assessed: (a) the number of awakenings with difficulty returning to sleep; (b) a 1–7 rating scale assessing restedness; (c) a 1–7 rating scale assessing difficulty falling asleep;

(d) the number of hours of sleep per night. The data were collected by means of a self-report sleep questionnaire (Monroe, 1968). The questionnaires were returned to the therapist's secretary each week prior to the weekly sessions. Scores for each subject were averaged for each week of the study.

Spouse–Roommate reliability checks. During the initial interview, clients were advised that participation in therapy required their spouse or roommate, where this was possible, to make a reliability check on the client's latency to sleep onset on at least one night of the study. Clients and the spouse–roommate were given instructions in reliability monitoring. The roommate–spouse was instructed to monitor the subject's sleep behavior by checking the following criteria: (a) eyes closed; (b) no voluntary movement for 10 min; and (c) no response from the client after the spouse–roommate whispers the client's name. The spouse–roommate received a separate questionnaire on which to record the number of minutes it took the client to fall asleep. To improve the accuracy of the reliability check the clients signed a witnessed contract prior to therapy agreeing that the client and the spouse–roommate would provide data in as accurate a manner as possible and that there would be no collusion.

PROCEDURE

Phase 1

Initially, clients proceeded through an intake interview with an independent assessor to obtain both historical and current details of the sleep problem and related behavior (e.g. drug usage). Additionally, subjects were acquainted with various aspects of the therapy program including appropriate self-monitoring of sleep behavior. Clients were informed that therapy would begin in 10 days allowing for the accumulation of sufficient baseline data to evaluate the therapy.

Phase 2

Clients met with the therapist for eight 45-min weekly sessions. The stimulus control condition was initiated with clients 1, 2 and 3 who were instructed to comply with the following regimen: (a) go to bed only when sleepy and do not attempt to obtain more sleep by retiring early; (b) do not read, watch TV, or eat in bed; (c) if you find yourself unable to fall asleep after 10 min, get up immediately and go do something else, and return to bed when sleepy; (d) set your alarm and get up at the same time every morning irrespective of how much sleep you received during the night; and (e) do not nap during the day. The remainder of the therapy sessions were spent helping the client to adhere as closely as possible to these instructions.

Simultaneously clients 4, 5 and 6 were provided with placebo instructions. The placebo control procedure, attributable to Steinmark and Borkovec (1974), was employed in the present study. This 'quasi-desensitization' condition required clients to construct an 18-item hierarchy of chronological bedtime activities to be paired with six neutral images. During the first session, clients in the placebo condition were required to construct the hierarchy and to develop six neutral images. During the remainder of the first therapy session and during weeks 3, 4 and 5 clients were instructed to intersperse the hierarchy items with the neutral images.

Phase 3

During weeks 6–9 clients 1, 2 and 3 (initially administered the stimulus control procedure) were maintained in stimulus control. Clients 4, 5 and 6 (initially receiving placebo instructions) were introduced to stimulus control procedure beginning with week 6 and were maintained in the condition through week 9.

On the week following the last treatment session, clients completed a Post-therapy Evaluation Questionnaire. All items were to be rated on a seven-point scale with "one" indicating a low score and "seven" denoting a high score. The following items were

included in this questionnaire:

1. How logical did this type of treatment seem to you?
2. Did you feel that the therapist was warm and accepting of your problems?

RESULTS AND DISCUSSION

Figure 1 depicts the results of the three phases of the study with regard to sleep-onset latency. Clients 1–3 administered stimulus control instructions during weeks 2–5 demonstrated reductions in sleep-onset latency to below 30 min. Clients 4–6 placed in the placebo condition following baseline obtained modest reductions in latency to sleep onset during weeks 2–5. However, clients 5 and 6 achieved clinically significant reductions in latency to sleep onset following transfer to the stimulus control condition during weeks 6–9. Figure 1 corroborates this description.

During initial sessions clients were given information on medication and side effects of rapid withdrawal and cautioned not to reduce medication intake abruptly. Clients were advised to become proficient with the procedure and then to gradually discontinue medication if they felt comfortable about undertaking such a reduction. The therapist retained a neutral stance with respect to medication. Interestingly clients 1, 2 and 3 reduced the dosage of sleep medication after exposure to the stimulus control therapy. However, virtually no changes in dosage level were observed with clients exposed to the placebo regimen. This suggests the possibility that clients who received the active

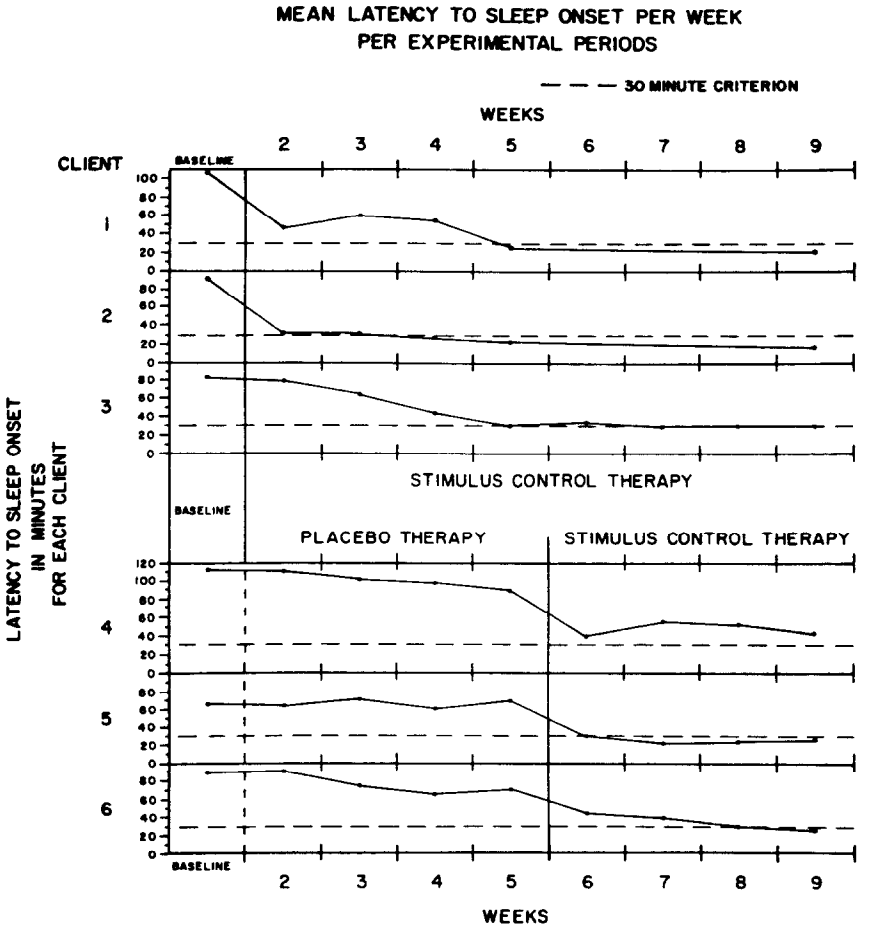

Fig. 1. Mean latency to sleep onset per week for the 9 weeks of the study is shown for the 6 clients. Following baseline clients 1, 2 and 3 were placed in the stimulus control condition; in contrast clients 3, 4 and 5 were placed in the placebo condition. At week 6 clients 3, 4 and 5 were placed in the stimulus control condition while clients 1, 2 and 3 were maintained in the stimulus control therapy.

therapy procedure were sufficiently confident in their ability to gradually reduce sedative–hypnotic intake. On the other hand, the placebo treatment apparently did not produce this same effect. Only when clients 4, 5 and 6 were introduced to stimulus control instructions did they reduce their medication intake.

In three of six cases, medication intake was eliminated following the stimulus control procedure. For the remaining three clients (2, 3 and 4) there were dramatic reductions in dosage, and client 2 shifted to a drug involving fewer complications. The changes in the ancillary dependent variables generally support the attribution of a positive, and efficacious, relationship between stimulus control instructions and a reduction in insomnia symptomatology. Specifically, clients reported feeling more well rested and less fatigued during the day.

All clients gave both stimulus control therapy and the placebo procedure credibility ratings of 6 or 7 on a 1–7 scale. Thus clients viewed the two procedures as equally credible.

The ratings of therapist warmth and contact were equal for all clients. Each client gave the therapist the highest possible, a 7 on the 1–7 scale. Consequently, differential expectancy of improvement due to treatment rationales or client–therapist interaction factors appear not to provide a strong alternative explanation for the outcome of the study.

The spouse–roommate reliability check data showed a marked agreement between the client's rating and the spouse or roommate's rating of latency to sleep onset. The spouse–roommate's estimates were all within 5 min of the clients' rating of the time required to fall asleep.

While several researchers have demonstrated that the stimulus control procedure ameliorates sleep disturbance to a magnitude statistically different from both no-treatment and placebo controls, the present study additionally indicates the stimulus control procedure can help clients to achieve clinically significant reductions in latency to sleep onset, sedative–hypnotic intake and a number of other self-report measures of insomnia symptomatology. With one exception, all clients required less than 30 min to fall asleep at the termination of therapy; while client 4 did not achieve this level, she did experience a two-thirds reduction in sleep-onset latency. It is possible that this client did not reach the 30 min criterion because she drastically reduced the dosage level of glutethimide during the last two weeks of the study. It is well known that marked withdrawal symptoms (one of which is delayed sleep-onset latency), accompany the reduction of sedative agents.

Further, in contrast to other studies mentioned above, a demonstration of the efficacy of stimulus control occurred in the context of very serious sleep disturbance.

As an initial attempt to assess the clinical utility of stimulus control therapy with severe insomniacs, the results of the present study are promising. Bootzin (1972) originally hypothesized that stimulus control therapy effected an improvement in maladaptive sleep patterns by restoring the bed as a discriminative stimulus for sleep. Zwart and Lisman (1976), however, obtained evidence suggesting that by rigidly controlling the time to retire and awaken a reduction in insomnia symptomatology will result. Zwart and Lisman hypothesized that stimulus control might work by interfering with arousal at bedtime. The clients in the present study made suggestions which support this possibility. All six clients pointed out that the stimulus control instructions served to break up lying in bed and thinking behavior. Many working adults appear to use the first minutes after retiring to ponder over the day's activities and to plan strategies for the next day. For the clients reported here, this reflection and problem solving activity became habitual, and out of the individual's control. Clients reported that the stimulus control instructions seemed to provide them with a means of asserting self-control over their bedtime cognitions by simply getting out of bed. The clients were very enthusiastic about this new-found self-control because they had come for therapy with the desire to become less dependent upon sleeping medication for rest and to regain the ability to view sleep as a natural process over which they could exert some voluntary

control. Consequently, their anxiety about the sleep problem diminished. Thus, the effectiveness of stimulus control may result from the diminution of anxiety concerning sleep loss which produces changes in sleep onset latency as Zwart and Lisman have hypothesized. This hypothesis, of course, requires additional data. In any event, the results of research into non-drug alternatives for the treatment of insomnia is promising and perhaps will soon provide methods which will be aids in reducing the widespread use of and dependence upon sedative–hypnotic agents.

REFERENCES

AMA DEPARTMENT OF DRUGS (1977) AMA drug evaluation (3rd Edn.). Publishing Sciences Group, Littleton, Massachusetts.

ASCHER M. and EFRAN J. (1978) The use of paradoxical intention in cases of delayed sleep-onset insomnia. *J. consult. clin. Psychol.* **8**, 547–550.

BOOTZIN R. (1972) Stimulus control treatment for insomnia. *Am. Psychol. Ass. Proc.* 395–396.

BOOTZIN R. and NICASSIO P. (in press) Behavioral treatments for insomnia. In *Progress in Behavior Modification* (Eds. M. HERSEN and R. M. EISLER). Academic Press, New York.

BORKOVEC T. and FOWLES D. (1973) Controlled investigation of the effects of progressive and hypnotic relaxation on insomnia. *J. abnorm. Psychol.* **83**, 153–158.

BORKOVEC T., KALOUPEK D. G. and SLAMA K. M. (1975) The facilitative effect of muscle tension-release in the relaxation treatment of sleep disturbance. *Behav. Ther.* **6**, 301–309.

BORKOVEC T., STEINMARK S. W. and NAU S. D. (1973) Relaxation training and single-item desensitization in the group treatment of insomnia. *J. Behav. Ther. exp. Psychiat.* **4**, 401–403.

BROWN J. S. (1965) Generalization and discrimination. In *Stimulus Generalization* (Ed. D. I. MOSTOFSKY). Stanford University Press, Stanford.

HAYNES S. N., PRICE M. G. and SIMONS J. B. (1975) Stimulus control treatment of insomnia. *J. Behav. Ther. exp. Psychiat.* **6**, 279–282.

HERSEN M. and BARLOW D. H. (1976) *Single Case Experimental Designs.* Pergamon Press, New York.

LAWRENCE P. S. and TOKARZ T. P. (1976) A comparison of relaxation training and stimulus control treatments for insomnia. Paper presented at the 10th Annual Meeting of the Association for the Advancement of Behavior Therapy, New York.

MONROE L. J. (1967) Psychological and physiological differences between good and poor sleepers. *J. abnorm. Psychol.* **72**, 225–264.

NICASSIO P. and BOOTZIN R. (1974) A comparison of progressive relaxation and autogenic training as treatments for insomnia. *J. abnorm. Psychol.* **83**, 253–260.

SLAMA K. (1975) Unpublished master's thesis, University of Iowa.

STEINMARK S. W. and BORKOVEC T. D. (1974) Active and placebo treatment effects on moderate insomnia under counter-demand and positive-demand instructions. *J. abnorm. Psychol.* **83**, 157–163.

TOKARZ T. P. and LAWRENCE P. S. (1974) An analysis of temporal and stimulus factors in the treatment of insomnia. Paper presented at the 8th Annual Meeting of the Association for the Advancement of Behavior Therapy, Chicago.

TURNER R. M. and ASCHER L. M. (1978) A methodological and statistical analysis of behavior therapy treatments for insomnia. Paper presented at the 49th Annual Meeting of the Eastern Psychological Association, Washington, D.C.

WILLIAMS R. L. and KARACAN I. (Eds.) (1975) *Pharmacology of Sleep.* John Wiley, New York.

WOOLFOLK R. L., CARR-KAFFASHAN and MCNULTY T. F. (1976) Meditation training as a treatment for insomnia. *Behav. Ther.* **7**, 359–365.

ZWART C. A. and LISMAN S. A. (1976) An analysis of stimulus control treatment for sleep-onset insomnia. Paper presented at the 10th Annual Meeting of the Association for the Advancement of Behavior Therapy, New York.